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CASE REPORT

Diabetic muscle infarction

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Introduction

Although diabetes mellitus is a very common disease, diabetic skeletal muscle infarction (DMI) is seldom reported in the literature, being a rare complication of poorly controlled diabetes. Therefore DMI is frequently clinically misdiagnosed as necrotizing fascitiis, cellulitis, abscess, neoplasm or myositis, often requiring biopsy.[1] We present our experience with DMI, in order to call attention to its clinical and radiological presentations and to help the radiologist to suggest the correct diagnosis.

Case report

A 55-year-old woman with a 27-year history of non-insulin dependent diabetes mellitus noted intense pain and swelling in her right calf and medial thigh 2 months before presentation. She had poorly controlled diabetes, with nephropathy treated by peritoneal dialysis. Her pain and swelling progressed over the next 4 weeks, resulting in a request for evaluation at our institution. On presentation both her thigh and calf were tender and swollen. She had a white blood cell count of $9.8\times10^3/mm^3$ and a mildly elevated creatine kinase (CK) of 6.7 ng/ml.

Plain radiographs of both legs were negative, but whole-body bone scintigraphy showed soft tissue uptake in both lower extremities (Fig. 1).

Magnetic resonance imaging (MRI) of both lower extremities (Fig. 2) with a 1.5-T unit showed swelling and increased signal intensity on T2-weighted images of the posterior muscle groups of both thighs and calves, the subcutaneous fat, and the fascia. T1-weighted images demonstrated asymmetry in the muscle bulk and strands of low-signal intensity in the subcutaneous fat. Gadolinium infusion led to diffuse enhancement of the involved muscles and fasciae.

Rhabdomyolysis or inflammation by a chemical irritant was excluded by normalization of the CK values (from 6.7 to 3.5 ng/ml) and absence of leakage of toxic materials contained in the dialysis solution on human serum albumin (HSA)-tagged peritoneal radiography.

An open biopsy specimen of involved muscle and fascia revealed areas of liquefaction necrosis and fibrosis without evidence of microangiopathy (Fig. 3). No microorganisms or parasites were identified. With appropriate analgesia, the woman's symptoms and laboratory values returned to baseline over the next month.

Discussion

Skeletal muscle infarction is an uncommon complication of poorly controlled diabetes mellitus when advanced microvascular and neuropathological complications have developed.[2] It tends to occur in type 1 insulin-dependent diabetes. All reported cases have had a history of at least 5 years of diabetes.[3] The cause of DMI is believed to be extensive thrombosis of medium and small arterioles; embolic thrombosis is believed to be a less likely cause.[1,4] People usually present with abrupt onset of severe pain in the anterior thigh and exquisitely tender swelling, in the absence of trauma. DMI is frequently bilateral.[4] The quadriceps are most frequently involved, followed by the thigh

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Figure 1 Whole-body bone scintigraphy shows soft tissue uptakes in both lower extremities.

adductors and hamstrings, and calf muscle involvement has also been reported.[1]

Unless there is a concurrent infection, these patients are usually afebrile, with a normal or mildly elevated white blood cell count without regional adenopathy. Their CK values are typically normal or mildly elevated; however, this may be related to the amount of time that has elapsed since the onset of symptoms.[1]

On histopathological examination, DMI may be seen as varying areas of muscle infarction with zonal necrosis, foci of haemorrhage, and fatty infiltration. Small arterioles show hyalinosis and thickening of the lumen. Small areas of haemorrhagic necrosis are surrounded by muscle fibres in various stages of degeneration and regeneration. Atherosclerotic calcifications may be seen within the medium-sized arteries.[4,5] In our case, biopsy of involved muscle and fascia revealed areas of liquefaction necrosis with fibrosis. We assumed that the liquefaction necrosis was the result of the ischaemia or anoxic damage caused by the several weeks of secondary inflammation.

MRI can define the anatomical location and extent of the pathological process and can exclude bone marrow involvement.[2,6] The infarcted muscle groups are seen as muscle swelling that is either isointense or hypointense on T1-weighted images and hyperintense compared with skeletal muscle on T2-weighted images.[1–4,6] Perifascial and subcutaneous oedema can also be seen, but are rather uncommon,[3] although they do reflect the acute oedema as well as the inflammatory changes.[1–4,6]

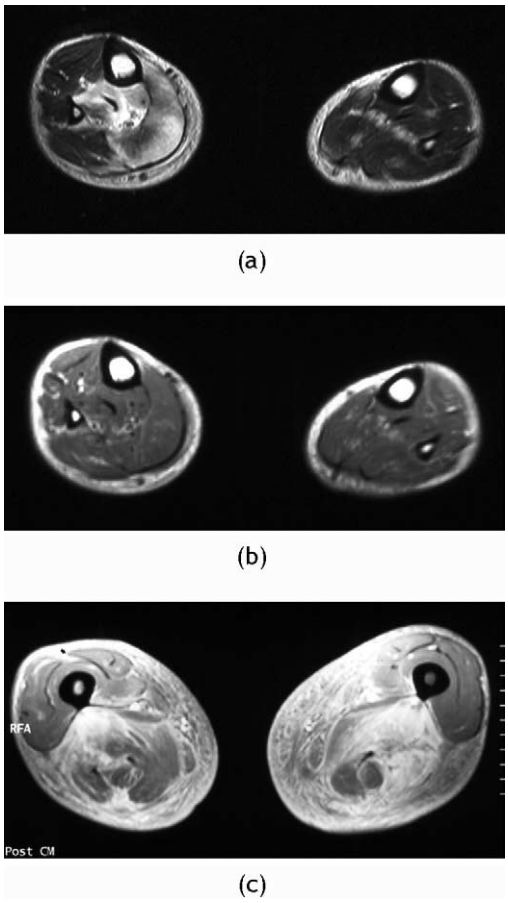

Figure 2 Magnetic resonance examination of both lower extremities with a 1.5-T unit shows (a) swelling and increased signal intensity on T2-weighted image of the posterior muscle groups of both thighs and calves, the subcutaneous fat, and the fascia. (b) T1-weighted image demonstrates asymmetry in the muscle bulk and strands of low-signal intensity in the subcutaneous fat. (c) Gadolinium infusion leads to diffuse enhancement of the involved muscles and fasciae.

The pattern of enhancement on gadolinium-enhanced T1-weighted images is diffuse and has small foci showing rim enhancement; these represent areas of actual muscle infarction and necrosis.[3,4] In our case, both infarcted muscles and subcutaneous fat were diffusely swollen and isointense on T1-weighted images, hyperintense on T2- weighted images, and appeared as diffuse enhancement with focal rim enhancement on gadolinium-enhanced images. The deep fasciae were also thickened and enhanced.

The differential diagnosis of diabetic muscle infarction includes cellulitis, soft tissue abscess, pyomyositis, necrotizing fasciitis, other causes of myositis (e.g. dermatomyositis, focal myositis, nodular myositis and proliferative myositis), and rhabdomyolysis.[1,4,7] This woman had a history of drug-taking which could have induced

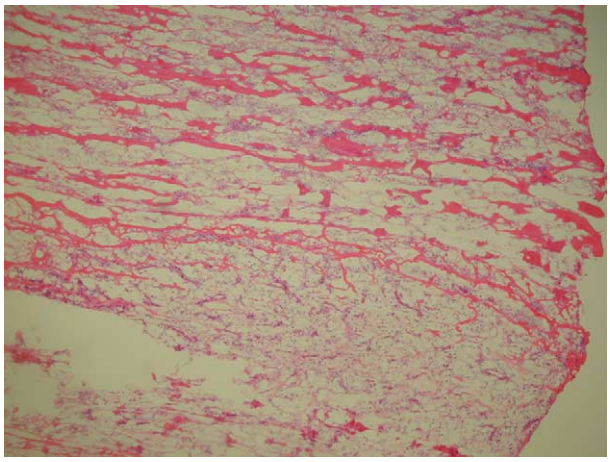

Figure 3 Specimen (haematoxylin and eosin, ×100) of involved muscle and fascia reveals areas of liquefaction necrosis and fibrosis without evidence of microangiopathy.

rhabdomyolysis and deteriorated renal function as seen at admission. Therefore, the differential diagnosis of rhabdomyolysis and DMI was difficult to make if based on clinical manifestations only. Because of laboratory findings with mild CK elevation with rapid normalization, negative myoglobinuria, and the MRI of diffuse subcutaneous infiltration more than muscle involvement, we were able to exclude rhabdomyolysis.[8,9] Toxic drug-induced chemical inflammation was also excluded because of the absence of leakage on HSA-tagged peritoneal radiography.

Necrotizing fasciitis is a major differential consideration that may not be distinguishable from DMI on the basis of MRI alone. However, because of the absence of fever, the presence of severe pain, and the multiple muscle involvement, we could easily exclude necrotizing fasciitis.

DMI is suspected in a patient with long-standing and poorly controlled diabetes when there is abrupt onset of severe leg pain with MRI showing diffuse oedema and swelling of multiple muscles, often in more than one compartment.[4] Radiologists should be familiar with this entity and may then suggest the correct diagnosis, in line with characteristic clinical presentation and imaging studies.

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